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Predicting epidemic size and probability in networks with heterogeneous infectivity and susceptibility

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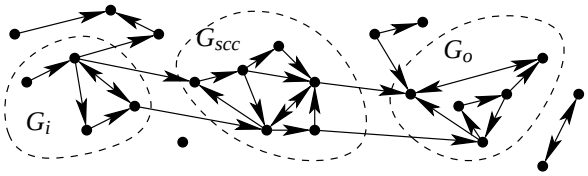
Introduction

Infectious diseases spread along human, animal, plant, or computer networks. Understanding properties of these networks gives insight into analyzing how the disease spreads, and so recent work has investigated the impact of network properties on epidemic spread. [1, 3, 4]. Most studies assume an average *transmissibility* (the probability that an infection of node i will result in infection of the neighboring node j), but it is well-known that the transmissibility is heterogeneously distributed. In this study we investigate the impact of this heterogeneity and derive rigorous bounds on the size and probability of epidemics for given average transmissibilities. The bounds we find give insight into optimal strategies to prevent or reduce the impact of epidemics.

We study epidemics spreading on large random networks, with fixed degree distribution. The nodes are divided into three classes:

- *Susceptible*: Nodes which may become infected if a neighbor is infected.
- *Infected*: Nodes which are infected and may infect susceptible neighbors.
- *Recovered*: Nodes which have been infected but are no longer. These nodes may not infect or be infected.

The infectiousness of an individual depends on a number of properties such as the levels of virus shedding or whether an employer allows sick days. Similarly the susceptibility of an individual depends on vaccination or exposure history and personal protective measures. The transmissibility T_{uv} from node u to v is $T(\mathcal{I}_u, \mathcal{S}_v)$ where



Schematic representation of G_i, G_{scc}, and G_o. All nodes in G_{scc} can reach any other node in G_{scc}.

$\mathcal{I}_u$ and $\mathcal{S}_v$ represent all factors affecting the infectiousness of u and susceptibility of v.

A standard approach to epidemic modeling is to take a single infected individual u (the *index case*) and consider its neighbors. Each neighbor v is infected with probability T_{uv}. The index case then recovers. We then consider the newly infected nodes and their susceptible neighbors, repeating until no infected nodes remain.

An equivalent approach is more computationally intense, but provides a useful theoretical framework. We consider each node u separately and determine *a priori* whether u would infect its neighbor v if u becomes infected while v is susceptible. If so, we place a directed edge from u to v. The edges of the original network are either lost or replaced with a directed edge, which may be bidirected as in figure 1. The index case is then chosen, and all nodes in its out-component are infected. The size of the outbreak is equal to the out-component size. Given large enough average transmissibility some nodes have an out-component limited in size only by the size of the network. We define these large outbreaks to be *epidemics*. In this case, G_i (the giant in-component), G_{scc} (the giant strongly-connected component) and G_o (the giant out component) exist as shown in figure 1. Epidemics occur if the initial infection is in either G_i or G_{scc}. The probability $\mathcal{P}$ of an epidemic is the fraction of nodes in $G_i \cup G_{scc}$, while at leading order the attack rate $\mathcal{A}$ (the fraction infected) is the fraction of nodes in $G_{scc} \cup G_o$. We note that interchanging the direction of the arrows interchanges G_i with G_o, so any method which calculates the probability of an epidemic can also calculate the size.

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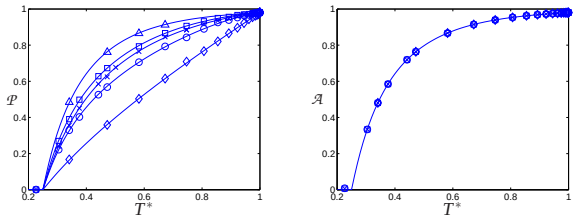
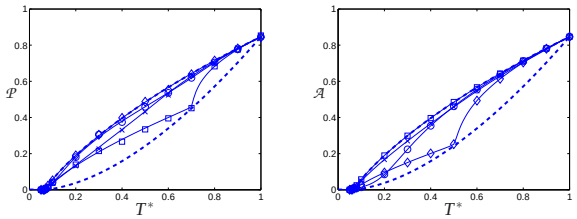


Comparison of theory (lines) with simulation (symbols) for an Erdős–Rényi network. With fixed infection rate, we set the recovery time to be $\tau = 5$ *(*$\triangle$*),* $\tau = 0$ *or* ∞ *(*$\Diamond$*),* $\tau = 2$ *or* 8 *(*$\square$*),* $\tau = 1$ *or* 10 *(*$\circ$*), and finally a constant recovery rate (*$\times$*).* $\mathcal{P}$ *changes, but* $\mathcal{A}$ *does not.*



Comparison of theory (curves) with simulation (symbols) for $T_{uv} = 1 - \exp(-\alpha I_u S_v)$ *in a scale-free network with a cutoff at high degree. The theoretical bounds are in dashed bold. The distributions are* $\Diamond$*:* $P(I) = \delta(I-1)$, $P(S) = 0.5\delta(S - 0.001) + 0.5\delta(S-1)$*;* $\times$*:* $P(I) = 0.5\delta(I-0.1) + 0.5\delta(I-1)$, $P(S) = 0.2\delta(S-0.1) + 0.8\delta(S-1)$*;* $\circ$*:* $P(I) = 0.5\delta(I-0.1) + 0.5\delta(I-1)$, $P(S) = 0.8\delta(S-0.011) + 0.2\delta(S-1)$*;* $\square$*:* $P(I) = 0.3\delta(I-0.001) + 0.7\delta(I-1)$, $P(S) = \delta(S-1)$.

Calculating Epidemic Probability

The random networks we study have few short loops, and so early in the outbreak we may assume it spreads on an infinite tree. We use generating functions [5] which encode a distribution of non-negative integers k a function by $f(x) = \sum_{k=0}^{\infty} p_k x^k$. We calculate a generating function $f(x,g)$ for the number of infections in generation g. The probability the epidemic dies out by generation g is $f(0,g)$. In the limit of infinite system size, $\mathcal{P} = 1 - \lim_{g\to\infty} f(0,g)$.

The detailed analysis is in [2]. We find rigorous upper and lower bounds on $\mathcal{P}$ in terms of the average transmissibility, T_0. For a fixed value of T_0, $\mathcal{P}$ is maximized if all nodes have probability T_0 to infect a random neighbor. Conversely, $\mathcal{P}$ is minimized if a fraction T_0 of the nodes infect all their neighbors while the remaining nodes infect none. Equivalent statements hold for susceptibility and $\mathcal{A}$. We demonstrate our results for Erdős–Rényi and scale-free networks in the figures.

Discussion

The bounds derived in [2] show that maximizing the variance in infectiousness for given average transmissibility minimizes an epidemic's probability. Similarly maximizing the variance in susceptibility minimizes its size. Consider two strategies with the same average effect, but one has heterogeneous impact on infectiousness (*e.g.*, incomplete contact tracing) while the other has heterogeneous impact on susceptibility (*e.g.*, incomplete vaccination). Which is optimal depends on whether an outbreak is established or not. If we seek to eliminate a rare disease, contact-tracing outperforms vaccination. In contrast, if we hope to reduce the impact of a widespread disease, vaccination outperforms contact tracing.

Acknowledgements

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